Amendment to the specification:

Please amend the specification under the provisions of 37 C.F.R. §1.121 as follows:

Please replace the paragraph starting at page 103, line 5, with the following amended paragraph:

> Antibody to the Her-2/neu gene product has been shown to inhibit the growth of breast cancer cells overexpressing Her-2/neu and to have clinical utility in treating breast cancer. We studied a recombinant, humanized anti-Her-2/neu antibody (~~Herceptin~~ the product Trastuzumab sold under the trademark HERCEPTIN) in preclinical models of human prostate cancer. The androgen-dependent CWR22 and LNCaP human prostate cancer xenograft models and androgen-independent sublines of CWR22 were used. Her-2/neu staining of the parental, androgen-dependent, and androgen-independent CWR22 tumors and LNCaP tumors demonstrated variable Her-2/neu expression. ~~Herceptin~~ HERCEPTIN was administered i.p. at a dose of 20mg/kg twice weekly after the xenograft had been established. No effect of ~~Herceptin~~ HERCEPTIN on tumor growth was observed in any of the androgen-independent tumors; however, significant growth inhibition was observed in both of the androgen-dependent xenograft models, CWR22 (68% growth inhibition at the completion of the experiment; P=0.03 for trajectories of the average tumor volume of the groups) and LNCaP (89% growth inhibition; P=0.002). There was a significant increase in prostate-specific antigen (PSA) index (ng PSA/ml serum/mm3 tumor) in ~~Herceptin~~ HERCEPTIN-treated androgen-dependent groups compared with control (CWR22, 18-fold relative to pretreatment value versus 1.0-fold, P=0.0001; LNCaP, 2.35-

fold relative to pretreatment value versus 0.6-fold, P=0.001). When paclitaxel (6.25mg/kg s.c., five times/week) was given to animals with androgen-dependent and independent tumors, there was growth inhibition in each group. Paclitaxel and ~~Herceptin~~ HERCEPTIN cotreatment led to greater growth inhibition than was seen for the agents individually. Thus, in these prostate cancer model systems, ~~Herceptin~~ HERCEPTIN alone has clinical activity only in the androgen-dependent tumor and has at least an additive effect on growth, in combination with paclitaxel, in both androgen-dependent and androgen-independent tumors. Response to ~~Herceptin~~ HERCEPTIN did not correlate with the PSA levels, because the PSA index markedly increased in the ~~Herceptin~~ HERCEPTIN-treated group, whereas it remained constant in the control group. These results suggest the utility of ~~Herceptin~~ HERCEPTIN in the treatment of human prostate cancer.